SUBCUTANEOUS TESTOSTERONE IMPLANT THERAPY IMPROVES ENDOTHELIAL-DEPENDENT AND INDEPENDENT VASODILATION IN POSTMENOPAUSAL WOMEN ALREADY RECEIVING OESTROGEN

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The gender difference in cardiovascular disease has been partly attributed to higher androgenic hormone levels. Although testosterone in women may not affect lipids, it remains unknown whether it negates favourable oestrogenic effects on endothelial function. We have investigated the effects of testosterone implant therapy on endothelial function (flow mediated vasodilation (FMD)) in women using hormone replacement therapy (HRT). B-mode ultrasound measurements of resting brachial artery diameter, following reactive hyperaemia (endothelium-dependent) and following glyceryl trinitrate (GTN) (endothelium-independent) dilation were recorded in 33 postmenopausal women stabilised on HRT (> 6 months), at baseline and 6 weeks after a testosterone implant (50mg), with 15 postmenopausal non-users of HRT serving as controls. In the brachial artery baseline resting diameter was similar (0.40 ± 0.04 cm) in all groups. The treated group, testosterone levels increased (0.99 ± 0.08 to 4.99 ± 0.3 mmol/L, p < 0.001), associated with a mean 42% increase in FMD (6.4% ± 0.7 to 9.1% ± 1.1, p = 0.03). The control group did not change (8.1% ± 1.4 to 5.6% ± 1.0, p = 0.4). There was significantly greater improvement in FMD in the testosterone-treated compared to control group (p = 0.04). GTN induced vasodilatation increased with testosterone treatment (14.9% ± 0.9 to 17.8% ± 1.2, p = 0.03).

Conclusion: Exogenous testosterone implants improve both endothelial dependent (flow mediated) and endothelium-independent (GTN mediated) brachial artery vasodilation in postmenopausal women, using long-term oestrogen therapy. The mechanisms underlying these potentially beneficial cardiovascular effects require further investigation.

LIPOCORTIN PROTECTS CARDIAC MYOCYTES FROM ISCHAEMIA IN VITRO.


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